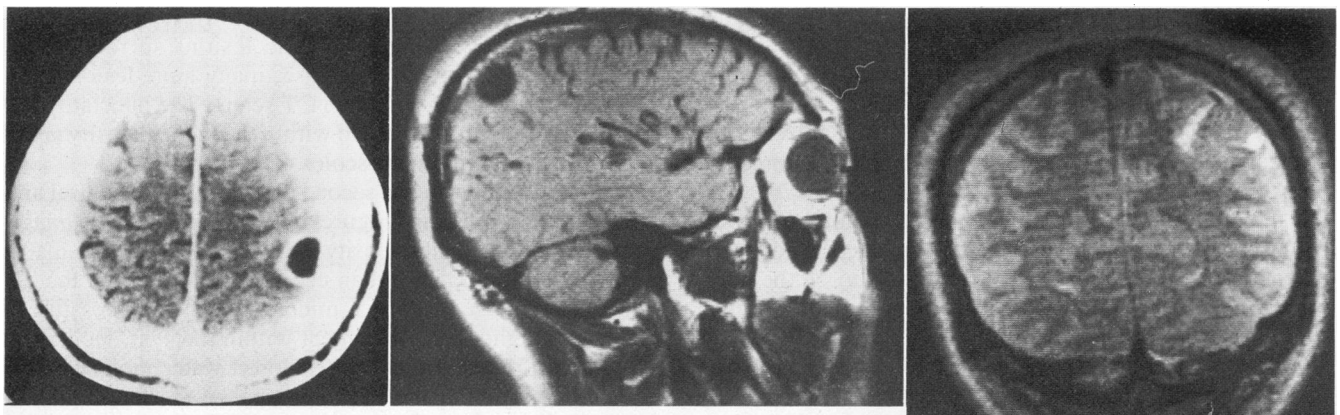

Figure 5.—Patient 2. **Left,** A rim-enhancing, rounded lesion is seen in the left parietal convexity on this contrast-enhanced computed tomographic scan. **Middle,** Sagittal (repetition time [TR] 850 ms, echo-delay time [TE] 40 ms) and, **right,** coronal (TR 2,000 ms, TE 80 ms) spin-echo images of the cysticercal cyst show a long T1 and medium-length T2, with the surrounding high intensity on the coronal scan probably representing edema. Effacement of the surrounding sulci is better shown on the T1-weighted image **(middle)**, while effacement of the normal surrounding white matter shows better on the T2-weighted coronal scan **(right)**.

for a nucleus in a magnetic field. They measure how quickly the nuclei return to their ground state energy level after excitation by a radio-frequency pulse. A short T1 relaxation time and a long T2 relaxation time are associated with high signal intensity on spin-echo sequences.[11])

The differential diagnosis in a patient with many long T1 and long T2 lesions in the brain includes multiple infarction (bland or septic), encephalitis subcorticalis chronica (Binswanger's disease), demyelinating disease and some metastases, as well as cysticercosis. The largely gray matter-subarachnoid space location of the lesions in our case would eliminate Binswanger's and demyelinating disease, and the clinical presentation should separate metastases and multiple infarcts from the presence of cysticercosis. Additionally, none of the above-mentioned diseases should present with a central, punctate, low-intensity focus.

Another feature that may be important in the lesions observed in our first case is their shape. As mentioned earlier, cysticerci tend to conform to the shape of the space they occupy when they are intraventricular or in the subarachnoid space. In case 1, the borders of the lesion tend to conform to those of the surrounding sulci. Unfortunately, no strongly T2-weighted imaging was done and, with the available images, it is not possible to separate cyst from surrounding edema. Possibly no cyst was present in these lesions, as none was seen on CT. If this is the case, we are probably just seeing edematous parenchyma. In future cases, additional spin-echo sequences will be used in an attempt to identify the cyst itself and determine its shape and T1 characteristics.

In our first case, follow-up MR imaging showed almost complete resolution of the lesions one month following therapy (Figure 4). Thus, another potential use for MR, especially in cases where the diagnosis is not completely firm, is to assure that appropriate response occurs following therapy.

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Hemophilus influenzae Pericarditis in Two Adults

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SERIOUS INFECTIONS caused by *Hemophilus influenzae* type b commonly occur during childhood. *H influenzae* has been recognized with increasing frequency in adults as a cause of

(Weingarten S, Weinberg H, Fang M, et al: *Hemophilus influenzae* pericarditis in two adults. West J Med 1986 Nov; 145:690-694)

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serious infections, including sinusitis, epiglottitis, bronchitis, pneumonia and, less commonly, meningitis, appendicitis, urogenital infections, cellulitis, cholecystitis, septic arthritis and endocarditis.[1] *H influenzae* pericarditis is uncommon; 15 cases of *H influenzae* pericarditis have been reported in adults, including two involving ampicillin-resistant strains.[2,3] We report two cases of *H influenzae* pericarditis, including one with pericardial empyema, in previously healthy adults.* Moreover, the first case describes identification of a β-lactamase-producing, ampicillin-resistant strain in a pregnant woman.

Reports of Cases

Case 1

The patient, a 37-year-old gravida 2, para 1, pregnant woman, was admitted because of one day of sore throat, right otalgia, nonproductive cough and anterior pleuritic chest pain. The patient had previously been in excellent health. There was no illness in the family or history of recent travel. She worked as a counselor for emotionally disturbed children who were about 8 years old; earaches and presumed ear infections had been common among them. Penicillin was given orally before admission. On admission, the blood pressure was 110/70 mm of mercury, heart rate 136 per minute, respiratory rate 28 per minute and temperature 37.8°C (100°F) by mouth. On physical examination she had slight pharyngeal erythema, right submandibular lymphadenopathy, crackles over the lower posterior thorax bilaterally and a fundal height of 32 cm, consistent with a 33-week gestation. The fetal heart rate was 170 per minute.

The leukocyte count was 18,800 per μl, with 22% neutrophils, 63% bands, 4% lymphocytes, 3% monocytes, 4% metamyelocytes and 4% myelocytes. A serum creatinine level and results of a urinalysis were normal. The admission chest radiograph showed a patchy left lower lobe interstitial infiltrate and a small right lower lobe infiltrate. The electrocardiogram revealed sinus tachycardia with a rate of 140 per minute. Gram's stain of a sputum specimen showed moderate polymorphonuclear leukocytes, Gram-positive cocci, Gram-negative cocci and Gram-negative rods. Blood cultures were done.

The patient was initially treated with ampicillin, 4 grams given intravenously per 24 hours, and subsequently erythromycin, 2 grams given intravenously per 24 hours, was added. Over the next 48 hours, the patient had increasing tachypnea and delirium and a metabolic acidosis developed. A repeat chest radiograph done 24 hours after admission showed small bilateral pleural effusions. On day 3, a thoracentesis of the left side of the chest yielded brown purulent fluid containing 215,000 leukocytes per μl, 99% of which were neutrophils. The fluid had a pH of 6.35, glucose 2 mg per dl, protein 3.6 grams per dl and lactic dehydrogenase (LDH) 2,898 units per liter. Gram's stain showed pleomorphic Gram-negative coccobacilli. A thoracentesis of the right side of the chest yielded similar findings. Bilateral closed chest tubes were placed and antibiotic therapy was changed to intravenous administration of ampicillin and gentamicin sulfate.

On cardiac monitoring the new onset of rapid atrial fibrillation was noted on day 4. A chest radiograph showed a newly enlarged cardiac silhouette and a two-dimensional echocardiogram revealed large anterior and posterior pericardial effusions. The patient underwent a left anterior thoracotomy and anterior pericardiectomy. Brown purulent pericardial fluid was obtained that had 168,000 leukocytes per μl with 90% neutrophils and a glucose concentration of 3 mg per dl, LDH 5,236 units per liter and protein 3.86 grams per dl. Gram's stain showed pleomorphic Gram-negative coccobacilli. A computed tomographic scan of the head and lumbar puncture showed no abnormalities. Cultures of blood and sputum specimens taken at admission and pleural and pericardial cultures grew *H influenzae* type b, biotype I, β-lactamase producing. Antibiotic therapy was changed to cefuroxime, 6 grams given intravenously per 24 hours. The patient temporarily required an endotracheal tube for acute respiratory failure. A serum immunoglobulin (Ig) G level was 347 mg per dl (normal 639 to 1,349) and IgA 47 mg per dl (normal 70 to 312). The patient was treated with a short course of γ globulin given intramuscularly. By the tenth hospital day, chest and endotracheal tubes were removed.

On the 14th hospital day, the patient gave birth to a 2,090-gram girl. There was no evidence of *H influenzae* infection in the baby or the placenta. Results of quantitative immunoglobulin studies done in the baby were normal. Results of repeat quantitative immunoglobulin studies of the patient during the convalescent period were normal, IgG level 1,190 mg per dl, IgA level 160 mg per dl and IgM level 209 mg per dl (normal 56 to 209). The patient and baby had an uneventual recovery.

Case 2

A 57-year-old woman was admitted because of dyspnea and a sore throat. She had previously been in excellent health. After her symptoms failed to improve on penicillin therapy given orally, she was admitted to hospital for further evaluation. On admission, the blood pressure was 100/70 mm of mercury, heart rate 112 per minute, respirations 48 per minute and temperature 35°C (95°F) by mouth. On physical examination she had pharyngeal erythema and thyromegaly. The leukocyte count was 14,400 per μl with 77% neutrophils, 13% lymphocytes and 10% monocytes. The admission chest radiograph showed bilateral infiltrates with pleural effusions. An electrocardiogram revealed a new right bundle branch block.

Initially the patient was treated with intravenous administration of erythromycin, nafcillin sodium and tobramycin. A thoracentesis was done and yielded 200 ml purulent fluid that contained 85,000 leukocytes per μl. The pH was 7.2 and a Gram's stain showed pleomorphic Gram-negative rods. Culture of pleural fluid grew *H influenzae*. Antibiotic therapy was changed to ampicillin and chloramphenicol given intravenously.

A repeat chest radiograph showed a globular cardiac silhouette. A repeat electrocardiogram revealed low-voltage, premature atrial contractions and ST-segment elevation consistent with pericarditis. A two-dimensional echocardiogram showed a moderate pericardial effusion. The patient had a new onset of atrial fibrillation, for which she was given digoxin and procainamide hydrochloride. She required an endotracheal tube for acute respiratory failure and administration of dopamine hydrochloride for hypotension.

*Marshall Kadner, MD, Stephen Uman, MD, and Bernard Axelrod, MD, gave permission to report these cases.

On the ninth hospital day, the patient underwent left thoracotomy and partial decortication. Her recovery was uneventful and three years later she remains in good health.

Comment

Purulent pericarditis is a serious disease with a fulminant course. The disease may be seen in association with pulmonary or intracardiac infection. Purulent pericarditis may also result from bacteremia or postoperative infection.[4] It occurs with increased frequency in debilitated patients, and fever, chills, night sweats and dyspnea are symptoms that usually accompany the infection. The mortality rate may be as high as 77%.[4] Organisms commonly associated include *Streptococcus pneumoniae, Streptococcus pyogenes* group A, *Staphylococcus aureus* and, less commonly, Gram-negative enteric organisms and fungi.[4,5]

H influenzae type b is an infrequent cause of purulent pericarditis in adults. The 15 previously reported cases of pericarditis associated with *H influenzae* are listed in Table 1. In only eight cases was the organism isolated from pericardial fluid. The ages of the 15 patients ranged from 18 to 60 years; ten were female. Most were previously healthy adults who presented initially with upper respiratory tract signs and symptoms. Ten patients presented with a sore throat, and ten had pneumonia or empyema associated with the pericarditis. Only one patient had *H influenzae* pericarditis without evidence of concurrent infection elsewhere. Blood cultures grew *H influenzae* in ten cases. All but three patients underwent pericardial drainage. There was evidence suggestive of *H influenzae* infection on Gram's stain of sputum, pleural or pericardial fluid in eight patients before bacteriologic confirmation. Two cases of ampicillin-resistant *H influenzae* purulent pericarditis were reported, with one shown to involve a β-lactamase-producing strain.[2] The mortality rate from *H influenzae* purulent pericarditis is significantly less than what is reported overall for purulent pericarditis.[4,5] Only 2 of 15 patients died of the illness. One death was related to sulfonamide-induced thrombocytopenia.[6]

The two patients presented here share many characteristics with previously reported cases. Both patients were previously healthy and presented with a severe sore throat and lower respiratory tract symptoms followed by clinical deterioration. The first patient, in particular, had early clinical features typical of adults with humoral immune deficiency in whom segmental pulmonary parenchymal disease develops.[16] Both patients were noted first to have *H influenzae* pneumonia and thoracic empyema. Purulent pericarditis ensued, most likely by continuous spread, although hematogenous spread cannot be excluded. In the first case, β-lactamase-producing *H influenzae* was cultured from the pericardial fluid.

In both cases, interpretation of the Gram's stain may have led to an early diagnosis of *H influenzae* infection before bacteriologic confirmation. A sputum Gram's stain was characteristic in 23% to 75% of adults with *H influenzae* pneumonia and bacteremia and in 71% of adults with nonbacteremic *H influenzae* pneumonia in previously reported series.[17,18]

H influenzae was once uniformly susceptible to ampicillin. Since the initial reports of ampicillin resistance in 1974[19] and then chloramphenicol resistance,[20] antibiotic resistance has become an increasing problem in treating cases of systemic bacterial infections. The prevalence of ampicillin resistance among *H influenzae* pediatric meningitis organisms rose from 19% in 1978 to 25% of the organisms reported more recently nationally with wide geographic variation.[21] The rates of resistance are lower in organisms identified from the respiratory tract. Resistance to chloramphenicol, especially among *H influenzae* type b bacilli, has remained low, with a rate of less than 1% in the United States.[22] A recent report from Spain noted that 41 of 225 strains of *H influenzae* were resistant to both ampicillin and chloramphenicol.[23] There is anecdotal experience with combined ampicillin and chloramphenicol resistance that is plasmid-borne in US isolates. At our hospital, currently 10% of *H influenzae* organisms are resistant to ampicillin, whereas no chloramphenicol-resistant organisms have been noted (M. A. Morgan, MD, unpublished data, Jan to Dec 1985).

Resistance to ampicillin in *H influenzae* is principally governed by the plasmid-mediated production of the triethylenemelamine β-lactamase enzyme[24] and, to a lesser degree, intrinsic resistance. Plasmid-mediated resistance to chloramphenicol is conferred via the production of chloramphenicol acetyltransferase. Another mechanism for ampicillin resistance is non-β-lactamase-mediated resistance due to alterations in penicillin-binding membrane proteins.[25] Additional factors implicated with antibiotic resistance include the following: previous β-lactam antibiotic therapy, geographic location and serotypes a to f (capsular antigen) and biotypes I to V classification.[26]

The initial choice of antimicrobial therapy is crucial in treating systemic infection in *H influenzae* and must include consideration of possible resistance. Antibiotic susceptibility testing and β-lactamase screening of all *H influenzae* organisms should be determined rapidly to guide subsequent appropriate therapy. One recommendation is that all cases of possible systemic infections due to *H influenzae* should initially be treated with chloramphenicol given intravenously at doses of 75 to 100 mg per kg every six hours (total maximum daily dose 4 to 6 grams a day) plus the addition of ampicillin at a dose of 150 mg per kg per day. If the organism is shown to be susceptible to ampicillin, the chloramphenicol regimen may be discontinued. The appropriate duration of antibiotic therapy depends on the site and extent of disease. The introduction of new cephalosporin antibiotics that penetrate cerebrospinal fluid, such as cefuroxime, cefotaxime sodium, moxalactam disodium, ceftizoxime sodium, ceftazidime, that have documented efficacy in the treatment of *H influenzae* systemic infections, including meningitis, offers possible advantages over either brief or prolonged courses of chloramphenicol therapy. Cefuroxime and selected "second"- and "third"-generation cephalosporins appear to be promising alternatives to chloramphenicol in treating life-threatening infections where β-lactamase-positive organisms are known or suspected.[27,28] The major drawbacks of these newer cephalosporins are the expense of some of the agents, the need to use ampicillin with them initially in empirical therapy for meningitis if *Listeria monocytogenes* or group B streptococci are considerations and the theoretic potential for the development of further *H influenzae* resistance.

The first case dealt with *H influenzae* type b, β-lactamase producing, in a pregnant patient who responded rapidly to the administration of cefuroxime. Chloramphenicol therapy was

TABLE 1.—Hemophilus Influenzae *Purulent Pericarditis in Adults*

Reference	Patient	Age (Years)	Sex	Symptoms	Ampicillin Resistance	Associated Infections	Positive Gram's Stain	Positive Cultures	Antimicrobial Treatment	Surgery	Outcome
Hensler, 1955[6]	1	24	♀	Cough, dyspnea and abdominal pain	...	Pneumonia and empyema	...	Pericardial fluid	Penicillin, sulfonamide, tetracycline	Pericardiotomy	Died
Crossley et al, 1973[7]	2	38	♀	Sore throat and chest pain	...	Pneumonia	Pleural and pericardial fluid	Blood, sputum, pleural and pericardial fluid	Cephalothin sodium, ampicillin, streptomycin	Pericardiectomy	Lived
Crossley et al, 1973[7]	3	44	♂	Sore throat and delirium	...	Pneumonia	Pleural fluid	Blood, throat and sputum	Ampicillin, kanamycin sulfate, chloramphenicol	Pericardiectomy	Lived
Duke and Donovan, 1973[3]	4	33	♀	Sore throat and neck tenderness	Yes	Pharyngitis	...	Pericardial fluid	Penicillin, cephalothin, ampicillin, chloramphenicol, dimethylchlor-tetracycline	Pericardiectomy	Lived
Alsever et al, 1974[8]	5	26	♀	Sore throat, fever, dysphagia and hoarseness	...	Thyroiditis, pneumonia, and empyema	Pleural and pericardial fluid	Blood, pleural and pericardial fluid	Cephalothin, chloramphenicol, ampicillin	Pericardiectomy	Lived
Rubin and Mollering, 1975[4]; Jafari et al, 1976[9]; Sink et al, 1982[10]	6	26	♀	...	...	Meningitis and upper respiratory tract infection	...	Pericardial fluid and blood	Chloramphenicol	Pericardiectomy	Lived
Jafari et al, 1976[9]	7	23	♀	Sore throat, vomiting, dyspnea and dysphagia	...	Empyema	Pericardial fluid	Pericardial and pleural fluid	Chloramphenicol	Pericardiectomy	Lived
Sink et al, 1982[10]	8	27	♂	Chest pain and dyspnea	...	None	Pericardial fluid	Pericardial fluid	Cefamandole, tobramycin, ampicillin	Pericardiectomy	Lived
Jennings et al, 1983[11]	9	59	♂	Chills and dyspnea	...	Pneumonia	Sputum and pericardial fluid	Blood and sputum	Tobramycin, penicillin, cefamandole, ampicillin	Pericardiectomy	Lived
Buckingham et al, 1983[12]	10	42	♀	Myalgias, sore throat, headache and neck stiffness	...	Pneumonia and meningitis	Pericardial fluid	Blood, sputum and pericardial fluid	Erythromycin, ampicillin	Pericardiectomy	Lived
Graham et al, 1983[13]	11	18	♂	Sore throat, chest pain and fever	...	Pneumonia	...	Blood	Gentamicin sulfate, cefazolin, ampicillin	None	Lived
Graham et al, 1983[13]	12	33	♂	Cough and chest pain	...	Pneumonia	...	Sputum and blood	Penicillin, oxacillin sodium, ampicillin	None	Lived
Mier and Shanson, 1984[2]	13	26	♀	Sore throat and swollen neck	β-Lactamase producing	Epiglottitis	...	Blood	Floxacillin, ampicillin, chloramphenicol	Pericardiectomy	Lived
Kiefaber et al, 1984[14]	14	31	♀	Sore throat and myalgias	...	Pneumonia	Pleural fluid	Pleural fluid and blood	Penicillin, ampicillin, chloramphenicol	Pericardiocentesis	Lived
Shiekh et al, 1981[15]	15	60	♀	Sore throat	...	Pharyngitis	...	Blood	...	None	Died
This report		37	♀	Sore throat, ear pain and chest pain	β-Lactamase producing	Empyema	Pleural and pericardial fluid	Blood, sputum, pleural and pericardial fluid	Ampicillin, erythromycin, cefuroxime, gentamicin	Pericardiectomy	Lived
This report		57	♀	Sore throat and dyspnea	...	Empyema	Pleural fluid	Pleural fluid	Erythromycin, nafcillin, tobramycin, ampicillin, chloramphenicol	None	Lived

avoided in this patient because of possible adverse effects on the fetus.[29] Biotype I of *H influenzae* identified from patient 1 has been described as more common than other biotypes in respiratory tract infections, although ampicillin resistance was not common in one study of biotype I organisms.[30]

An important complication of pericarditis is cardiac tamponade. Timely drainage either by surgical intervention or by means of repeated pericardiocentesis or catheter drainage is usually required for *H influenzae* pericarditis. Anterior interphrenic pericardiectomy has recently been recommended for drainage and prevention of constrictive pericarditis.[31] The patient in case 1 underwent pericardiectomy and had pronounced hemodynamic improvement following the procedure. Remarkably, our second patient and two previously reported patients had an uneventful recovery from their illness without drainage. None of these cases, however, had cultural confirmation of *H influenzae* purulent pericarditis. Aggressive diagnostic techniques and a drainage procedure are still recommended.

Although *H influenzae* is a rare cause of pericarditis in adults, it should be particularly considered in any patient presenting with an antecedent history of upper respiratory tract symptoms and a concurrent pneumonia, particularly with an associated empyema. Gram's stain of the sputum, pleural fluid or pericardial fluid (or all three) will often provide the diagnosis before culture confirmation; the use of counterimmunoelectrophoresis is another consideration. The prognosis is good with a combined approach of appropriate antimicrobial therapy that initially takes into account the prospect of bacterial resistance and adequate surgical drainage.

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Treatment of Cranial Osteomyelitis From Disseminated Coccidioidomycosis

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COCCIDIOIDOMYCOSIS has been recognized for less than 100 years with as many as 100,000 cases occurring yearly in the United States. The fungus is acquired by the respiratory route and usually causes only asymptomatic infection found by skin test conversion. It is estimated to account for 70 deaths annually.[1]

The fungus is endemic to areas of the southwestern United States and Central and South America. Within the past four decades studies have suggested the incidence and severity of infection is related to race,[2,3] immune competence,[4] pregnancy[5] and age.[3] Blacks and Filipinos, patients with defects of immune competence, pregnant women and persons at either extreme of age seem more likely to have development of the infection and complications associated with dissemination.

Report of a Case

This 13-month-old black male child was in good health until 10 months of age when a slowly enlarging subcutaneous mass developed in the right maxillary region and in the right foot. The child was a military dependent whose family had recently been stationed in the San Joaquin area of California.

(Gillespie R: Treatment of cranial osteomyelitis from disseminated coccidioidomycosis. West J Med 1986 Nov; 145:694-697)

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